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Original Article

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Evaluation of Long-Term Kidney Functions of Patients Developing Hospital-Associated Acute Kidney Injury During COVID-19 Infection

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ABSTRACT

Background: This study aimed to evaluate the long-term kidney outcomes of patients hospitalized with COVID-19 by comparing those who developed acute kidney injury (AKI) during hospitalization with those who did not.
Methods: Among 3472 patients hospitalized with COVID-19, 95 patients who developed AKI and had follow-up creatinine measurements for at least 3 months were included. A control group of 370 patients without AKI during hospitalization was also analyzed. Kidney function outcomes were evaluated using serum creatinine, estimated glomerular filtration rate (eGFR), and the incidence of chronic kidney disease (CKD) at follow-up.
Results: Patients who developed AKI were significantly older and had higher creatinine levels at admission. At a median follow-up of approximately 5 months, follow-up creatinine levels and delta creatinine were significantly higher in the AKI group compared with patients without AKI ($P < .001$). Correspondingly, eGFR declined more markedly in patients with AKI. Overall, CKD developed in 16.1% of patients at follow-up and was significantly more frequent among those who experienced AKI during hospitalization. In multivariate analysis, AKI remained an independent predictor of persistent kidney dysfunction.
Conclusion: The COVID-19 patients who develop AKI during hospitalization are at increased risk of persistent kidney dysfunction and CKD after discharge. These findings highlight the importance of long-term kidney monitoring in this patient population.
Keywords: Acute kidney injury, chronic kidney disease, Covid-19, creatinine

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INTRODUCTION

The COVID-19 infection started in China in December 2019 and became a pandemic within a few months. Although COVID-19 infection is seen as a disease with lung involvement in the foreground, it has been observed that kidney involvement (proteinuria, hematuria, acute kidney injury (AKI), glomerulonephritis) is quite common over time.

Acute kidney injury can develop especially in severe cases of COVID-19 infection. Based on a meta-analysis involving 20 cohorts, the incidence of AKI was found to be 17% and it was shown that the development of AKI increases the risk of mortality 4-15 times.[1] According to recent studies, besides the damage caused by the COVID-19 virus itself as the cause of kidney involvement, additional causes such as nephrotoxicity of the agents used in the treatment, hypovolemia, and co-infection have been identified. Several studies have identified acute tubular injury as the predominant etiology of AKI.[2] In a retrospective observational study examining patients during the early period following hospital discharge,

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it was shown that 46% of hospitalized patients developed AKI and 35% of the patients still could not achieve their basal functions at the time of discharge and 32% of the 212 patients who were followed up on the median 21st day after discharge from the hospital still did not achieve their basal creatinine levels.[3] This study was a retrospective, single-center investigation conducted in China, involving a limited number of patients; it was shown that approximately half of the patients who developed AKI in the first 3 weeks had a full recovery of kidney function.[4] However, this rate rises to 80% in the first 10 days in patients with acute kidney disease with other causes.[5] Advanced-stage AKI is associated with markedly increased mortality among hospitalized COVID-19 patients. Traditional risk factors for mortality in the general COVID-19 population, including older age, male sex, and comorbidities, are also significantly associated with in-hospital mortality in patients with AKI. Interestingly, preexisting non-dialysis chronic kidney disease (CKD) did not appear to elevate in-hospital mortality among AKI patients. Moreover, kidney dysfunction persisted in a substantial proportion of patients after hospital discharge.[6] A recent study reported that patients with COVID-19–related AKI showed greater post-discharge kidney function decline than those with non–COVID-19–related AKI.[7]

The pathophysiology of COVID-19–related AKI is multifactorial, involving systemic inflammation, endothelial injury, and activation of coagulation pathways. The SARS-CoV-2 may directly affect kidney tubular and endothelial cells via angiotensin-converting enzyme-2 receptors, while cytokine release, endothelial dysfunction, and microthrombi formation lead to kidney ischemia and tubular injury. In critically ill patients, additional factors such as hypoxia, hypotension, nephrotoxic drugs, and mechanical ventilation further aggravate kidney damage. Histopathological findings such as acute tubular injury, endotheliitis, and thrombotic microangiopathy support this multifactorial mechanism.

MAIN POINTS

- Acute kidney injury (AKI) during COVID-19 hospitalization is associated with persistent impairment of kidney function during long-term follow-up.
- Patients who developed AKI exhibited significantly higher follow-up creatinine levels and greater declines in estimated glomerular filtration rate (eGFR) compared with patients without AKI.
- Chronic kidney disease (CKD) (eGFR <60 mL/min/1.73 m^2) developed in a substantial proportion of patients after AKI, indicating incomplete kidney recovery in some cases.
- Lower baseline eGFR and the presence of AKI were independent predictors of CKD development during follow-up.
- These findings underscore the importance of structured long-term kidney monitoring in patients hospitalized with COVID-19, even in those without apparent AKI at presentation.

The long-term consequences of COVID-19–related AKI remain unclear. In this study, 3472 patients hospitalized for COVID-19 in the institution's pandemic units were screened, and 465 patients were included to compare long-term kidney outcomes between those who developed AKI and those who did not during hospitalization. Accordingly, the study aimed to assess the long-term impact of COVID-19 infection on kidney function in both groups.

MATERIALS AND METHOD

Ethics committee approval (Approval No: 2020-245, Date: 18.02.2021) was obtained Ethics Committee of Haseki Training and Research Hospital from for this single-center, retrospective observational study. A total of 3472 patients who were hospitalized with a confirmed diagnosis of COVID-19 between March 11, 2020, and October 1, 2020, in the pandemic units of the institution were screened. Verbal informed consent was obtained from all participants in accordance with ethical guidelines.

Acute kidney injury was defined according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours or ≥1.5 times from baseline within 7 days. Estimated glomerular filtration rate (eGFR) was calculated using the 2009 Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation without race coefficient, in accordance with recommendations for European populations.

Baseline creatinine was determined as the lowest serum creatinine value recorded within the 7 days prior to hospital admission or from pre-admission laboratory measurements when available. Delta creatinine was defined as the difference between the follow-up creatinine level measured at least 3 months after discharge and the minimum creatinine value recorded during hospitalization.

Patients admitted to the intensive care unit, those without available follow-up creatinine measurements at least 3 months after discharge, patients receiving chronic kidney replacement therapy before hospitalization, and patients who died during hospitalization or within the first 3 months after discharge were excluded from the study.

Among the 3472 screened patients, 548 patients showed a creatinine increase of at least 0.3 mg/dL during hospitalization. Forty-nine patients who were already receiving chronic hemodialysis prior to hospitalization were excluded. According to KDIGO criteria, 254 patients met the diagnostic criteria for AKI. Of these, 23 patients died during hospitalization and were excluded from further analysis. Among the remaining patients, 34 died during the post-discharge period and were therefore excluded. In addition, 18 patients required initiation of chronic hemodialysis following COVID-19–associated AKI and continued dialysis during follow-up. These patients were considered to have developed end-stage kidney disease and were included

in the CKD outcome analysis. Finally, 95 patients with AKI had available follow-up creatinine measurements at 3 months or later and were included in the AKI group.

For comparison, 370 patients without AKI during hospitalization who had available creatinine measurements at least 3 months after discharge were selected from the remaining hospitalized COVID-19 population and included as the control group (Figure 1). Matching for age, sex, or comorbidities was not performed; therefore, potential demographic and clinical differences between groups were considered as possible confounders.

The duration of follow-up was calculated as the time between hospital admission and the first available creatinine measurement obtained at least 3 months after discharge. Kidney function was assessed using both serum creatinine and eGFR. Changes in kidney function were evaluated by calculating delta creatinine and delta eGFR values between baseline and follow-up measurements.

Chronic kidney disease was defined as an eGFR <60 mL/min/1.73 m^2 at or beyond 3 months after discharge. The CKD severity was classified according to KDIGO eGFR stages (G3a-G5). Patients who required maintenance hemodialysis during follow-up were classified as having end-stage kidney disease.

Serum creatinine, urea, sodium, potassium, alanine aminotransferase (ALT), aspartate aminotransferase (AST), C-reactive protein (CRP), and ferritin levels were measured using the COBAS 8000 analyzer (Roche Diagnostics, Mannheim, Germany) in the biochemistry laboratory of the hospital. Lymphocyte and neutrophil counts were measured using the Mindray CAL 8000 hematology analyzer in the microbiology laboratory.

Statistic Analysis

The IBM SPSS Statistics for Windows 25.0 program (IBM SPSS Corp.; Armonk, NY, USA) was used for statistical analysis. Descriptive statistics were given as numbers and percentages for categorical variables, mean and standard deviation for data with normal distribution for numerical variables, and median

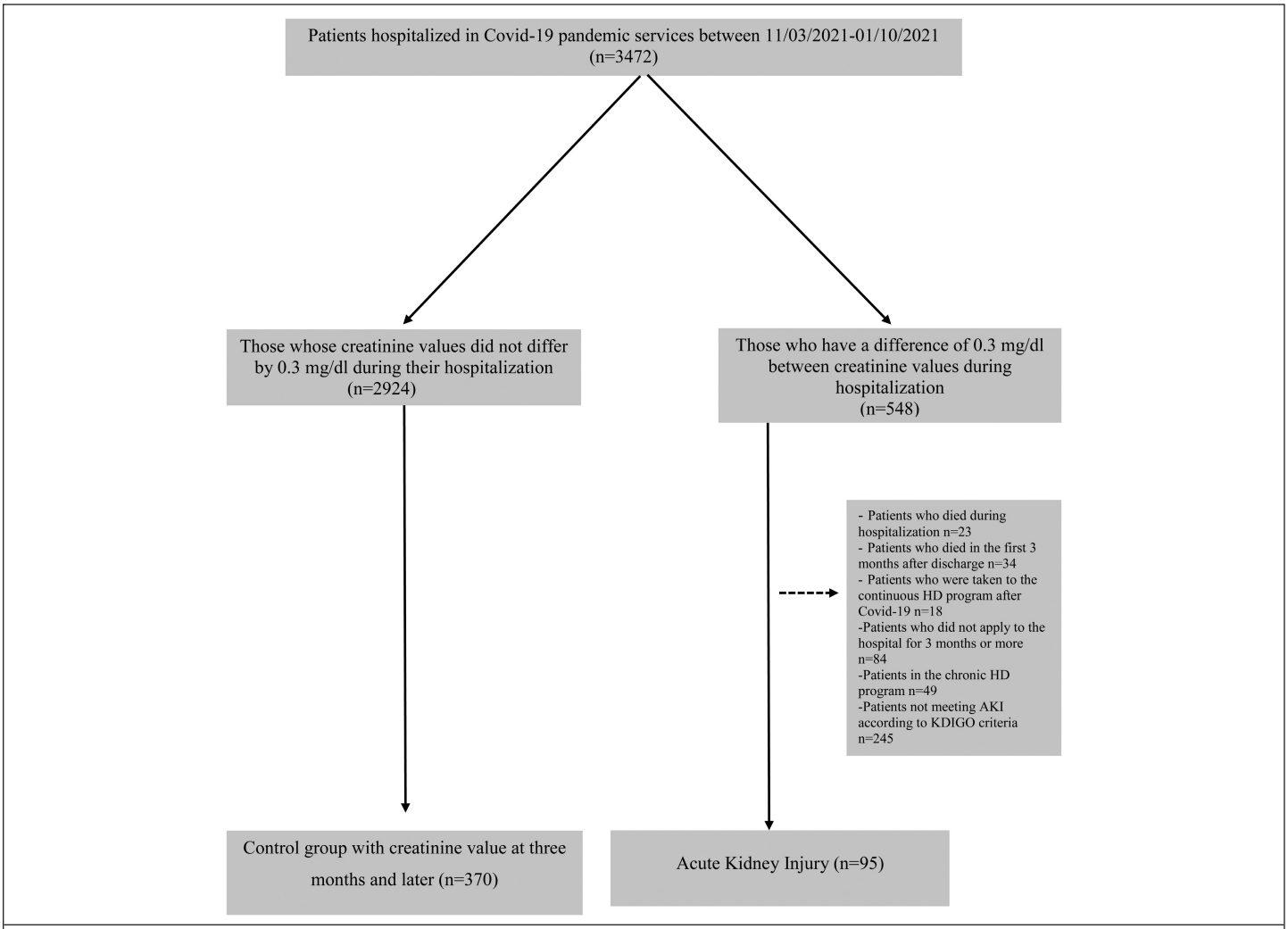

Figure 1. Flowchart of patient selection and grouping according to AKI status and follow-up criteria.

interquartile range (IQR) (25%-75%) for abnormal distributions. The normal distribution of data was analyzed with the Kolmogorov–Smirnov test. Chi-square test was used to compare the categorical variables of the 2 groups (with and without AKI). Mann–Whitney *U*-test was used to compare the non-normal distribution of the numerical variables of the 2 groups. Correlation analyses were performed with Spearman correlation analysis. Wilcoxon test was used to evaluate the comparison of non-parametric values before and after AKI injury. Multivariate analysis of factors determining delta creatinine was performed using linear regression analysis. Statistical alpha significance level was accepted as $P < .05$.

RESULTS

Among the 465 patients enrolled in the study, 53.5% were women and their mean age was 56.5 ± 14.5 years. The median follow-up of the study cohort was 142 days (IQR: 110-190). At admission, the median urea level was 29 mg/dL (IQR: 21-42), creatinine 0.73 mg/dL (IQR: 0.60-0.89), lymphocyte count 1.3 $\times 10^3/\mu L$ (IQR: 0.9-1.8), and CRP 19.5 mg/L (IQR: 8.0-52.5). Patients who developed AKI were followed for a median of 150 days (IQR: 110-217), compared with 139 days (IQR: 109-186) in those who did not develop AKI. When comparing 95 patients with AKI and 370 patients without AKI, those who developed AKI were significantly older. While their urea, creatinine, and procalcitonin levels at admission were higher in those with AKI, lymphocyte, neutrophil, CRP, fibrinogen, sodium, potassium, calcium, ALT, and AST levels were found to be lower (Table 1).

When the data of patients with and without AKI were examined, patients who developed AKI had significantly higher minimum and maximum creatinine levels, as well as higher follow-up creatinine and delta creatinine values measured at least 3 months after discharge, compared with the control group. Median ≥3-month follow-up creatinine levels were 1.07 mg/dL (IQR: 0.82-1.44) in patients with AKI, 0.78 mg/dL (IQR: 0.62-0.91) in patients without AKI, and 0.81 mg/dL (IQR: 0.65-0.98) in the overall cohort. Similarly, delta creatinine values were higher in patients with AKI [0.21 mg/dL (IQR: 0.07-0.37)] compared with those without AKI [0.11 mg/dL (IQR: 0.03-0.20)] and in the

Table 1. Demographic Characteristics of Patients With and Without Acute Kidney Injury and Laboratory Values at the Time of First Hospitalization

	All Patients (N = 465)	Without AKI (N = 370)	AKI (N = 95)	*P*
Demographic characteristics				
Age (Years), mean ± SD	56.5 ± 14.5	54.0 ± 14.2	66.4 ± 11.2	<.001
Gender, n/N (%)				.261
Female	249/465 (53.5)	203/370 (54.9)	46/95 (48.4)	
Male	216/465 (46.5)	167/370 (45.1)	49/95 (51.6)	
Laboratory values, median (IQR)				
Creatinine (mg/dL)	0.73 (0.60-0.89)	0.71 (0.58-0.86)	0.82 (0.65-1.13)	<.001
Urea (mg/dL)	29.0 (21.0-42.0)	26.0 (19.9-34.1)	56.0 (36.0-80.0)	<.001
Lymphocyte ($10^3/\mu L$)	1.3 (0.9-1.8)	1.48 (1.09-1.98)	0.82 (0.58-1.21)	<.001
Neutrophil ($10^3/\mu L$)	3.5 (2.4-4.7)	3.68 (2.61-5.08)	3.2 (2.2-4.1)	<.001
C-reactive protein (mg/L)	19.5 (8.0-52.5)	23.4 (9.7-60.5)	12.9 (4.3-25.4)	<.001
Erythrocyte sedimentation rate (mm/h)	40.0 (21.0-60.0)	39.0 (22.0-58.0)	50.5 (16.5-74.0)	.448
Ferritin (ng/mL)	108.0 (51.0-217.0)	102.3 (47.6-212.4)	131.4 (64.3-240.4)	.052
Fibrinogen (mg/dL)	394.0 (346.0-505.0)	406.5 (354.7-519.2)	365.0 (293.0-441.0)	<.001
D-dimer (mg/L)	0.59 (0.39-1.15)	0.59 (0.38-1.09)	0.68 (0.43-1.34)	.139
Alanine aminotransferase (U/L)	21.0 (15.0-34.0)	22.5 (16.0-36.0)	18.0 (11.0-27.0)	<.001
Aspartate aminotransferase (U/L)	28.0 (20.0-39.0)	29.0 (22.0-41.0)	23.0 (16.0-33.0)	<.001
Sodium (mEq/L)	138.0 (135.0-140.0)	138.0 (136.0-141.0)	134.0 (131.0-137.0)	<.001
Potassium (mEq/L)	4.1 (3.8-4.4)	4.1 (3.9-4.4)	3.7 (3.2-4.1)	<.001
Calcium (mg/dL)	8.7 (8.3-9.1)	8.9 (8.5-9.3)	7.95 (7.52-8.37)	<.001
Procalcitonin (ng/mL)	0.05 (0.03-0.08)	0.04 (0.03-0.07)	0.06 (0.04-0.09)	.002

AKI, acute kidney injury.

overall population [0.13 mg/dL (IQR: 0.04-0.23)]. Both follow-up creatinine and delta creatinine values were significantly higher in patients who developed AKI ($P < .001$).

Consistent with these findings, eGFR values calculated using the 2009 CKD-EPI equation without race coefficient demonstrated a significant decline in patients with AKI. Baseline median eGFR was lower in the AKI group and further decreased at follow-up. At ≥3 months, median eGFR was 61.5 mL/min/1.73 m^2 in patients with AKI compared with 96.7 mL/min/1.73 m^2 in patients without AKI ($P < .001$). Accordingly, the decline in kidney function, expressed as delta eGFR, was markedly greater in patients with AKI (−26.3 mL/min/1.73 m^2) than in those without AKI (−5.0 mL/min/1.73 m^2) ($P < .001$).

As a consequence of this persistent impairment in kidney function, CKD, defined as an eGFR <60 mL/min/1.73 m^2 at or beyond 3 months after discharge, developed in 75 patients (16.1%) in the overall cohort. The CKD occurred significantly more frequently in patients who developed AKI during hospitalization [50 patients (52.6%)] compared with those without AKI [25 patients (6.8%)] ($P < .001$). Among patients who developed CKD, 7.3% were classified as stage G3a, 7.1% as stage G3b, and 1.7% as stages G4-G5 according to KDIGO eGFR categories (Table 2).

When the patient groups were examined separately, the maximum creatinine levels of the patients who developed AKI were 1.5 mg/dL (IQR: 1.20-2.20), and the minimum creatinine levels were 0.82 mg/dL (IQR: 0.65-1.13) and the first creatinine levels were significantly higher than 0.82 mg/dL (IQR: 0.65-1.13). The follow-up creatinine values were 1.07 mg/dL (IQR: 0.82-1.44) and were higher than the minimum and first creatinine levels, but lower than the maximum creatinine values. In patients without AKI, initial creatinine levels were 0.71 mg/dL (IQR: 0.58-0.86), minimum creatinine levels were higher than 0.64 mg/dL (IQR: 0.52-0.78), while maximum creatinine levels were 0.75 mg/dL (IQR: 0.62-0.89), follow-up creatinine levels were lower than 0.78 mg/dL (IQR: 0.62-0.91). It was determined that the creatinine levels of the patients without AKI during their follow-up were higher than the maximum creatinine levels during the hospitalization. When all the patients were examined, the initial creatinine values of the patients were 0.73 mg/dL (IQR: 0.60-0.89) and the minimum creatinine values were higher than 0.67 mg/dL (IQR: 0.54-0.82) while the maximum creatinine values were lower than 0.82 mg/dL (IQR: 0.65-1.07) and follow-up creatinine levels were less than 0.81 mg/dL (IQR: 0.65-0.98). The follow-up creatinine levels of all patients were lower than the maximum creatinine levels developed during hospitalization (Table 3).

Incidence of CKD, defined as eGFR <60 mL/min/1.73 m^2 at ≥3 months after discharge, according to the presence of AKI during hospitalization. Chronic kidney disease occurred significantly more frequently in patients who developed AKI compared with those without AKI (52.6% vs. 6.8%, $P < .001$) (Figure 2).

Correlation analyses revealed that the delta creatinine in all patients was positively associated with age ($r = 0.126$, $P = .007$), maximum creatinine ($r = 0.126$, $P = .007$), urea ($r = 0.122$, $P = .008$), ferritin ($r = 0.154$, $P = .001$), procalcitonin ($r = 0.133$, $P = .005$), and creatinine at ≥3 months follow-up ($r = 0.493$, $P < .001$). In contrast, delta creatinine was negatively correlated with lymphocyte count ($r = −0.168$, $P < .001$), sodium ($r = −0.150$, $P = .001$), and calcium levels ($r = −0.203$, $P < .001$). Creatinine at ≥3 months follow-up was positively correlated with age ($r = 0.358$, $P < .001$), minimum creatinine ($r = 0.771$, $P < .001$), maximum creatinine ($r = 0.751$, $P < .001$), creatinine at admission ($r = 0.755$, $P < .001$), urea ($r = 0.751$, $P < .001$), D-dimer ($r = 0.101$, $P = .032$), ferritin ($r = 0.190$, $P < .001$), and procalcitonin ($r = 0.288$, $P < .001$), but negatively correlated with sodium ($r = −0.169$, $P < .001$) and calcium ($r = −0.202$, $P < .001$). Creatinine at admission was positively correlated with age ($r = 0.292$, $P < .001$), minimum creatinine ($r = 0.925$, $P < .001$), maximum creatinine ($r = 0.770$, $P < 0.001$), urea ($r = 0.559$, $P < .001$), ferritin ($r = 0.166$, $P < .001$), procalcitonin ($r = 0.322$, $P < .001$), potassium ($r = 0.144$, $P = .002$), and ≥3-month follow-up creatinine ($r = 0.755$, $P < .001$), whereas negative correlations were observed with sodium ($r = −0.131$, $P = .005$) and lymphocyte count ($r = −.172$, $P < 0.001$). No

Table 2. Comparison of Kidney Function Parameters During Hospitalization and Follow-up in Patients With and Without AKI

	All Patients (n = 465)	Without AKI (n = 370)	AKI (n = 95)	*P*
Minimum creatinine (mg/dL)	0.67 (0.54-0.82)	0.64 (0.52-0.78)	0.82 (0.65-1.13)	<.001
Maximum creatinine (mg/dL)	0.82 (0.65-1.07)	0.75 (0.62-0.89)	1.50 (1.20-2.20)	<.001
≥3-month creatinine (mg/dL)	0.81 (0.65-0.98)	0.78 (0.62-0.91)	1.07 (0.82-1.44)	<.001
Delta creatinine (mg/dL)	0.13 (0.04-0.23)	0.11 (0.03-0.20)	0.21 (0.07-0.37)	<.001
Baseline eGFR (mL/min/1.73 m^2)	98.4 (82.7-110.1)	101.7 (86.2-112.3)	87.8 (70.4-102.6)	<.001
≥3-month eGFR (mL/min/1.73 m^2)	92.8 (74.4-106.0)	96.7 (82.1-109.4)	61.5 (44.8-83.7)	<.001
Delta eGFR	−5.6	−5.0	−26.3	<.001
CKD at ≥3 months (eGFR <60), n (%)	75 (16.1%)	25 (6.8%)	50 (52.6%)	<.001

AKI, acute kidney injury; eGFR outcomes and development of chronic kidney disease.

Table 3. Comparison of the Creatinine Values at the Third Month and Later in All Patient Groups With the Creatinine Values at Admission and the Minimum and Maximum Creatinine Values During the Hospitalization

	Admission Creatinine (mg/dL)	Minimum Creatinine (mg/dL)	Maximum Creatinine (mg/dL)	≥3-month Creatinine (mg/dL)	*P*
AKI (n = 95)	0.82 (0.65-1.13)	0.82 (0.65-1.13)	1.50 (1.20-2.20)	1.07 (0.82-1.44)	.317[a]; <.001[bcdef]
Without AKI (n = 370)	0.71 (0.58-0.86)	0.64 (0.52-0.78)	0.75 (0.62-0.89)	0.78 (0.62-0.91)	<.001[abcde]; .035[f]
All patients (n = 465)	0.73 (0.60-0.89)	0.67 (0.54-0.82)	0.82 (0.65-1.07)	0.81 (0.65-0.98)	<.001[abcde]; .002[f]

AKI, acute kidney injury.
[a]admission creatinine and minimum creatinine
[b]admission creatinine and maximum creatinine
[c]admission creatinine and third month and after creatinine
[d]minimum creatinine and maximum creatinine
[e]minumum creatinine and third month and after creatinine
[f]maximum creatinine and third month and after creatinine

significant correlation was found between delta creatinine and minimum creatinine.

Multivariate linear regression analysis demonstrated that the presence of AKI and lower baseline eGFR were independently associated with higher delta creatinine levels. When multivariate analysis of factors affecting delta creatinine value was conducted, among the factors such as presence of AKI, age (year), D-Dimer (ng/mL), lymphocyte (10^3/µL), male gender, CRP (log) (mg/L), it was revealed that only AKI at admission and lower baseline eGFR were independently associated with the development of CKD at the third month.

AKI remained a strong predictor of CKD development after adjustment for age, sex, baseline kidney function, and inflammatory markers. (*P* =<.001, OR = 0.214, 95% CI 0.097-0.330) (Table 4).

DISCUSSION

In this study evaluating long-term kidney outcomes in hospitalized COVID-19 patients, individuals who developed AKI during hospitalization exhibited significantly worse kidney function at follow-up compared with those without AKI. Although partial recovery of kidney function occurred after discharge, creatinine levels remained higher and the decline in kidney function was more pronounced in the AKI group. In multivariate analysis, the development of AKI during hospitalization was identified as an independent predictor of persistent kidney dysfunction, as reflected by higher delta creatinine values during follow-up.

Previous studies conducted during the early phase of the COVID-19 pandemic demonstrated that AKI occurred more frequently among patients with COVID-19 compared with those hospitalized without COVID-19 (56.9% vs. 25.1%) and that these patients had higher dialysis requirements and lower recovery rates during hospitalization (42.3% vs. 68.5%).[9] Even before the COVID-19 pandemic, AKI had been recognized as an important risk factor for the development and progression of CKD.[9-11]However, because COVID-19 is a relatively new disease, the long-term kidney consequences of COVID-19–associated AKI remain incompletely understood.

Several studies have investigated early kidney recovery following COVID-19–related AKI. In a retrospective observational study, AKI developed in 46% of hospitalized patients, and 35%

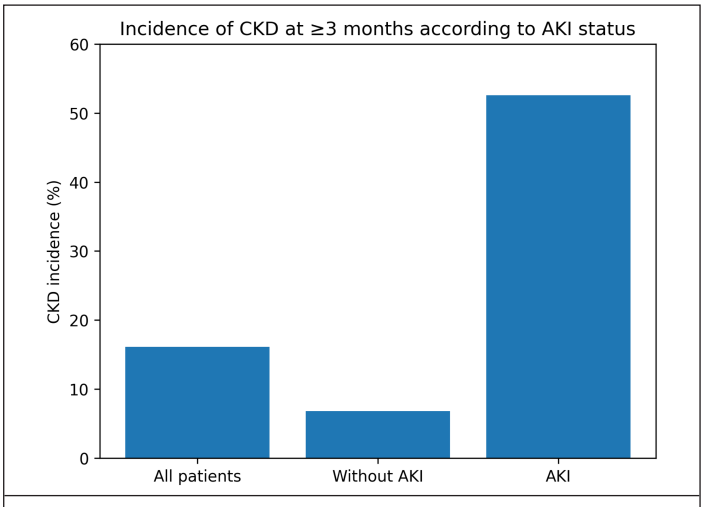

Figure 2. Incidence of chronic kidney disease at ≥3 months after discharge according to AKI status.

Table 4. Multivariate Linear Regression Analysis of Factors Associated With Delta Creatinine

Variable	β Coefficient	95% CI	*P*
AKI	0.214	0.097-0.330	<.001
Age (Years)	0.001	−0.002-0.004	.353
D-dimer (ng/mL)	0.002	−0.025-0.028	.908
Male (Sex)	0.073	−0.008-0.155	.078
Baseline eGFR	-0.92	−0.90-0.44	<.001
CRP (log)	0.007	−0.065-0.079	.852

N = 465; R^2 = 0.146; Adjusted R^2 = 0.132.
AKI, acute kidney injury; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; OR, odds ratio.

of these patients had not recovered their baseline kidney function at the time of discharge. Moreover, 32% of patients who were followed at a median of 21 days after discharge had not returned to their baseline creatinine levels.[3]Similarly, another retrospective single-center study from China reported that approximately half of the patients with AKI recovered their kidney function within the first 3 weeks.[4]In contrast, recovery rates are reported to be higher in AKI caused by other conditions, reaching up to 80% within the first 10 days.[5]

In a retrospective cohort study comparing 182 patients with COVID-19–associated AKI and 1430 patients with non-COVID-19–related AKI, the annual decline in eGFR among patients with COVID-19–associated AKI was 11.3 mL/min/1.73 m^2 greater than that observed in patients with non-COVID-19 AKI. This difference remained significant after adjustment for comorbidities and AKI severity. Furthermore, among patients whose kidney function had not recovered at discharge, those with COVID-19–related AKI demonstrated poorer recovery during outpatient follow-up compared with patients who developed AKI due to other causes.[7]

Importantly, the results demonstrate a markedly higher incidence of CKD among patients who developed AKI during COVID-19 hospitalization. CKD at ≥3 months occurred in 52.6% of patients in the AKI group compared with only 6.8% in patients without AKI, indicating a substantially increased risk of persistent kidney dysfunction following AKI. The pronounced decline in eGFR observed in the AKI group further supports the hypothesis that COVID-19–associated AKI may lead to sustained structural and functional kidney damage. These findings emphasize the importance of long-term kidney monitoring in patients who develop AKI during COVID-19 hospitalization.

In addition, the study demonstrated that serum creatinine levels at follow-up were significantly higher than the minimum creatinine values observed during hospitalization even among patients who did not develop AKI during their hospital stay. This finding suggests that kidney dysfunction may persist or emerge during the post-discharge period even in patients without overt AKI during the acute phase of COVID-19.

Despite the consistency between previous reports and our findings, several limitations should be considered. First, kidney function assessment relied solely on serum creatinine and creatinine-based eGFR calculations. Although these methods have recognized limitations, they remain the most widely used tools in clinical practice and epidemiological research because of their accessibility and feasibility. More precise methods for measuring kidney function are costly, labor-intensive, and difficult to implement in large patient cohorts.

Second, race information required for certain eGFR equations was unavailable in medical records; however, this limitation was addressed by applying the 2009 CKD-EPI equation without a race coefficient in accordance with current recommendations for European populations. Additionally, in critically ill and elderly patients, reduced muscle mass and fluid imbalance may further compromise the accuracy of serum creatinine as a surrogate marker of kidney function. Therefore, kidney function trends in the study were evaluated using both absolute creatinine values and changes in creatinine and eGFR over time.

Other limitations include the retrospective study design and the absence of urinary findings such as hematuria, leukocyturia, and proteinuria, as well as kidney imaging data. Nevertheless, since the primary objective of this study was to evaluate longitudinal changes in kidney function, the absence of these parameters is unlikely to have substantially affected the main conclusions. Furthermore, the control group was not matched for baseline characteristics such as age, sex, or comorbidities, and therefore residual confounding cannot be completely excluded.

Despite these limitations, the study has several important strengths. It was conducted in a tertiary referral hospital located in a densely populated metropolitan area and included a large screened population of 3472 patients, providing valuable real-world data on long-term kidney outcomes following COVID-19 hospitalization. These findings underscore the importance of post-discharge kidney follow-up even in patients without documented AKI during hospitalization and highlight the potential role of COVID-19 as a contributor to CKD development.

Patients who develop AKI during hospitalization for COVID-19 should be closely monitored because of the risk of incomplete kidney recovery and the potential development of CKD. In addition, long-term evaluation of kidney function may also be warranted in patients who did not develop AKI during hospitalization, as subtle kidney dysfunction may persist or emerge after recovery from COVID-19 infection.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Artificial Intelligence Usage Statement: This study used ChatGPT to improve language and grammar during the preparation of the manuscript. The authors take full responsibility for the content of the manuscript.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of the University of Health Sciences (Approval no: 2020-245, Date: 18.02.2021).

Informed Consent: This study used ChatGPT to improve language and grammar during the preparation of the manuscript. The authors take full responsibility for the content of the manuscript.

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